AMENDMENTS TO THE CLAIMS

Please amend the claims as follows:

This listing of claims will replace all prior versions, and listing, of claims in the application:

Listing of Claims:

Claim 1 (currently amended): A method for treatment of heart failure comprising attenuating PLB-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition and enhancing contractility in a heart comprising ~~inducing phospholamban deficiency, wherein~~

(a) providing a compound comprising an exogenous dominant negative phospholamban (PLB) protein functionally attached to a transport ~~penetratin~~ peptide; and

(b) contacting the heart with the compound, thereby attenuating PLB-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition and enhancing contractility in a heart to treat the heart failure ~~delivered to cardiac tissue induces phospholamban deficiency~~.

Claims 2 and 3 (canceled)

Claim 4 (currently amended): The method ~~for treatment of heart failure~~ of claim 19, wherein the mutations of PLB comprise sense ~~point~~ mutations.

Claims 5 to 11 (canceled)

Claim 12 (currently amended): A method for treatment of heart failure comprising enhancement of cardiac contractility by inhibition of phospholamban (PLB)-[[PLB-]] sarcoplasmic reticulum calcium ATPase (SERCA2a) interaction comprising

(a) providing ~~wherein~~ an exogenous dominant negative PLB protein functionally attached to a transport ~~penetratin~~ peptide; and

(b) delivering an effective amount of the compound ~~delivered~~ to cardiac tissue ~~is~~

~~used~~ to inhibit interaction between PLB and SERC2a, thereby enhancing cardiac contractility and treating heart failure.

Claims 13 to 15 (canceled)

Claim 16 (previously presented): The method of claim 22, wherein the mutations of PLB comprise sense ~~point~~ mutations of PLB.

Claims 17 and 18 (canceled)

Claim 19 (currently amended): The method ~~for treatment of heart failure~~ of claim 1, wherein the exogenous dominant negative PLB protein comprises a PLB protein with mutations.

Claim 20 (currently amended): The method ~~for treatment of heart failure~~ of claim 1, wherein the exogenous dominant negative PLB protein comprises a truncated PBL protein.

Claim 21 (canceled)

Claim 22 (currently amended): The method ~~for treatment of heart failure~~ of claim 12, wherein the exogenous dominant negative PLB protein comprises a PLB protein with mutations.

Claim 23 (currently amended): The method ~~for treatment of heart failure~~ of claim 12, wherein the exogenous dominant negative PLB protein comprises a truncated PBL protein.

Claim 24 (new): A method for attenuating phospholamban (PLB)-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition in a heart cell or a muscle cell, comprising

(a) providing a compound comprising an expression construct comprising a coding sequence for a dominant negative PLB functionally linked to a promoter active in the heart or the muscle cell, and the dominant negative PLB binds to wild-type PLB; and

(b) contacting the heart cell or the muscle cell with an effective amount of the compound, thereby attenuating phospholamban (PLB)-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition in the heart cell or the muscle cell.

Claim 25 (new): A method for increasing cardiac SR Ca^{2+} ATPase (SERCA2a) activity in a heart cell or a muscle cell,

(a) providing a compound comprising an expression construct comprising a coding sequence for a dominant negative phospholamban (PLB) functionally linked to a promoter active in the heart or the muscle cell, and the dominant negative PLB binds to wild-type PLB in the heart or the muscle cell; and

(b) contacting the heart cell or the muscle cell with an effective amount of the compound, thereby attenuating PLB-induced SERCA2a inhibition and increasing SERCA2a activity in the cell.

Claim 26 (new): The method of claim 24 or claim 25, wherein the heart cell is a cardiac myocyte.

Claim 27 (new): The method of claim 24 or claim 25, wherein the muscle cell is a smooth muscle cell.

Claim 28 (new): The method of claim 24 or claim 25, wherein the contacting is *in vitro*.

Claim 29 (new): The method of claim 24 or 25, wherein the contacting is *in vivo*.

Claim 30 (new): The method of claim 24 or 25, wherein the expression construct comprising a coding sequence for a dominant negative PLB further comprises a coding sequence for a penetratin peptide.

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Claim 31 (new): A method for enhancing contractility in a heart comprising

(a) providing a compound comprising an expression construct comprising an expression construct comprising a coding sequence for a dominant negative phospholamban (PLB) functionally linked to a promoter active in the heart, and the dominant negative PLB binds to wild-type PLB; and

(b) contacting the heart with an effective amount of the compound, thereby attenuating PLB-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition and enhancing contractility in the heart.

Claim 32 (new): A method for increasing heart activity comprising

(a) providing a compound comprising an expression construct comprising a coding sequence for a dominant negative phospholamban (PLB) functionally linked to a promoter active in the heart, and the dominant negative PLB binds to wild-type PLB; and

(b) contacting the heart with an effective amount of the compound, thereby attenuating PLB-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition and increasing heart activity.

Claim 33 (new): A method for treating heart failure comprising

(a) providing a compound comprising an expression construct comprising a coding sequence for a dominant negative phospholamban (PLB) functionally linked to a promoter active in the heart, and the dominant negative PLB binds to wild-type PLB; and

(b) contacting the heart with an effective amount of the compound, thereby attenuating PLB-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition, enhancing contractility or relaxation in the heart and treating the heart failure.

Claim 34 (new): The method of claim 31, claim 32 or claim 33, wherein the expression construct comprises an adenoviral expression construct.

Claim 35 (new): The method of claim 31, claim 32 or claim 33, wherein the

dominant negative phospholamban (PLB) has an altered amino acid sequence as compared to wild-type PLB.

Claim 36 (new): The method of claim 31, claim 32 or claim 33, wherein the dominant negative phospholamban (PLB) is a truncated PLB.

Claim 37 (new): The method of claim 36, wherein the truncated PLB comprises SEQ ID NO:8.

Claim 38 (new): The method of claim 36, wherein the truncated PLB comprises a PLB cytoplasmic domain.

Claim 39 (new): The method of claim 31, claim 32 or claim 33, wherein the dominant negative phospholamban (PLB) has an altered amino acid sequence as compared to wild-type PLB.

Claim 40 (new): The method of claim 1 or claim 12, wherein the exogenous dominant negative PLB protein is linked to the transport peptide by a covalent linkage.

Claim 41 (new): The method of claim 40, wherein the covalent linkage comprises a polylysine, a single peptide bond or a disulfide bond.

Claim 42 (new): The method of claim 41, wherein the polylysine comprises a branched polylysine.

Claim 43 (new): The method of claim 40, wherein the transport peptide comprises an antennapedia transport peptide or a penetratin.

Claim 44 (new): The method of claim 40, wherein the antennapedia transport peptide comprises SEQ ID NO:7.

Claim 45 (new): The method of claim 1 or claim 12, wherein the exogenous dominant negative PLB protein comprises the first 16 residues of SEQ ID NO:8; SEQ ID NO:17; SEQ ID NO:18; or SEQ ID NO:19.